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Association Between Lung Function and Exposure to Smoke Among Firefighters at Prescribed Burns

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We investigated the short-term effects of exposures to $PM_{3.5}$, acrolein, formaldehyde, and carbon monoxide on lung function in a group of firefighters performing prescribed burns. Spirometric measurements were made on 65 firefighters at the beginning, midpoint, and end of their work shift, while exposure was measured over the entire day. The interquartile range (IQR) of daily personal $PM_{3.5}$ exposures was 235 $\mu g/m^3$ to 1317 $\mu g/m^3$, with an average daily exposure of 882 $\mu g/m^3$. Concentrations of acrolein (IQR: [0.002, 0.018] ppm), formaldehyde (IQR: [0.008, 0.085] ppm), and carbon monoxide (IQR: [2.10, 10.48] ppm) were similarly elevated. In this group of firefighters, FEV_1 changed by −0.125 L from preshift to postshift ($p < .001$). We examined the association between this cross-shift lung function decrement and smoke exposure. A 1000 $\mu g/m^3$ increase in $PM_{3.5}$ was associated with a −0.030 L change in the cross-shift FEV_1 (95% CI [−0.087, 0.026]). Acrolein, formaldehyde, and carbon monoxide exposure were also not significantly associated with changes in FEV_1, FVC, or FEF_{25-75}. We concluded that while firefighters' lung function significantly decreased from preshift to postshift, firefighters exposed to greater concentrations of respiratory irritants did not experience greater lung function decrements. We could not establish a significant link to any of the individual toxic components of smoke we measured.

Keywords respiratory disease, particulate matter, formaldehyde, acrolein, carbon monoxide

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INTRODUCTION

Fine particulate matter is a major occupational and community pollutant that is often generated by mobile combustion sources such as trucks, buses, and automobiles. However, other sources vary geographically from coal fire power plants in the eastern United States to biomass burning in the western United States. Biomass burning includes wildfires, prescribed burning of forest and range, and agricultural burns. Smoke from this burning contains fine particles and carbon monoxide as well as a number of components known to be toxic to the human respiratory system.[1,2] These include nitrogen dioxide, aldehydes, and hazardous air pollutants such as benzo(a)pyrene.

Community exposure to various size fractions of particulate matter (PM) at 24-hour concentrations ranging from 30–110 $\mu g/m^3$ is associated with increased hospital admissions for asthma, increased emergency department visits for asthma, and increased mortality in individuals with pre-existing cardiac or respiratory disease.[3] Exposure to wood smoke from residential burning has been associated with respiratory infections and asthma aggravation in children.[1] Community exposure to smoke from wildland forest fires is associated with increased visits to hospital emergency departments.[4,5]

The Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) for respirable PM (50% collection efficiency at 3.5 μm, $PM_{3.5}$) in the workplace is 5 mg/m^3 according to Title 29, Part 1910.1000, Tables Z-1 and Z-3 of the *Code of Federal Regulations*. Carbon monoxide (CO) is another air pollutant that is emitted from fires. The OSHA occupational standard for CO presently is an 8-hr time-weighted average (TWA) of 50 ppm. However, some states enforce more stringent standards, such as the 35 or 25 ppm PELs with a 200 ppm short-term exposure limit established by Washington and California, respectively.

Several years ago, we studied pulmonary function values in 76 forest firefighters engaged in prescribed burning in western states. At that time, it was estimated that 70,000 prescribed fires occurred during each fire season.[6] Our study reported a significant decrement in forced expiratory volume in 1 sec (FEV_1) comparing pre-shift to postshift values.[7] Exposure to smoke by the subjects in this study also was measured; however, the relationship between smoke exposure and health effects was not explored at that time. This article describes the relationships between exposure and health data in 65 of the 76 firefighters in the previous study.

METHODS

The U.S. Forest Service measured smoke exposure at prescribed burns between 1991 and 1994 to determine the firefighters' average exposure during burns and over entire

work shifts, which include time traveling and setting up the burns. Firefighters at prescribed burns apply fire using torches and patrol the prescribed burn perimeter with hand tools and fire hoses to maintain the fire within the prescription boundaries. Prescribed burns averaged 7 hours in on-site work with potential exposure to combustion products. Firefighters have been found to have more exposure to smoke at prescribed burns than they experience during wildfire suppression.[8]

The study measured exposure to several inhalation compounds including respirable particulate matter ($PM_{3.5}$), CO, formaldehyde, and acrolein.[9] Respirable PM was measured by NIOSH method 0600.[10] Carbon monoxide was measured by nondispersive infrared spectrophotometry;[11] formaldehyde and acrolein were measured by sorbent cartridge with HPLC analysis according to EPA Method TO-11.[12] All firefighters were equipped with a backpack that contained personal sampling pumps, collecting consecutive air samples from the breathing zone during the work shift. Each sampled period generally lasted the duration of a given work activity. After the conclusion of a sample period, a technician would begin a new sample as quickly as possible if smoke exposure continued.

TWA concentrations were calculated for each firefighter to assess smoke exposure over the duration of a work shift and while on the fire line. Each TWA was calculated using the formula:

$$\mathrm{TWA} = (C_1 * T_1 + C_2 * T_2 + \cdots + C_N * T_N)/(T_1 + T_2 + \cdots + T_N) \quad \textbf{(1)}$$

where T_N is the time spent in period N and C_N the concentration in period N. Air pollution exposures during clean-air situations were estimated to be equivalent to background levels for the TWA calculation.

During the fall and spring of 1992 and 1993, lung function measurements were also gathered on 76 firefighters working the prescribed burns.[7] A single NIOSH-trained technician performed spirometry according to standards set forth by the American Thoracic Society. The technician used one spirometer in all tests and calibrated the equipment before and after each test to ensure standardization of measurements. When possible, field data were collected at three times during the day: immediately before, during, and immediately after a prescribed burn. Subjects completed a questionnaire to determine their smoking history, recent illness, allergies, and history of previous lung disease before being monitored. Current smokers were included among the sample of firefighters, but they were asked to refrain from smoking while the samples were being collected.

In the present study, we used linear regression to investigate the association between lung function decrements and pollution exposure for the 65 firefighters for whom the exposure assessment and lung function testing overlapped. We examined three measurements of lung function, FEV_1, FVC, and FEF_{25-75}, as well as four types of pollutants ($PM_{3.5}$, acrolein, formaldehyde, and CO). TWA concentrations of pollutants were compared to a firefighter's pre- to postshift change in lung function. We fit separate regression models using a single pollutant as the main predictor of interest in assessing lung function decrements. The pollutants were fit in separate models because they were highly correlated with each other and we wanted to assess their effects independently. We used multivariate models to adjust for gender, age, and current smoking status.

To assess the combined effects of exposure to the respiratory irritants acrolein, formaldehyde, and $PM_{3.5}$, we calculated a respiratory irritant exposure index (E_m). These three pollutants all cause irritant effects in the respiratory tract and mucous membranes. We calculated the E_m value using the American Conference of Governmental Industrial Hygienists' (ACGIH®) occupational guidelines for formaldehyde, acrolein, and $PM_{3.5}$[13] using the formula:

$$E_m = \frac{[\text{formaldehyde}]}{0.3} + \frac{[PM_{3.5}]}{3} + \frac{[\text{acrolein}]}{0.1} \quad \textbf{(2)}$$

An E_m level below 1 indicates that the combination of respiratory irritants is below the threshold limit value for the mixture of respiratory irritants.

RESULTS

Firefighters participating in this study were primarily white (93%) males (80%). Of the nonwhite subjects, 3% were Native Americans, 1% Native American–Caucasian mix, 1% Mexican Americans, and 1% African American–Caucasian mix. Eleven of the subjects were current smokers; 4 reported current lung problems that could affect pulmonary function tests (primarily upper respiratory infections), and 14 had present allergies. On average the subjects were 29 years of age, ranging from 19 to 56 years. One subject had an exceptional change in all lung function measurements, including an FEV_1 change of –1.69 L from pre- to postshift; all other subjects' FEV_1 change ranged from –0.65 L to 0.21 L. A 25-year-old male, noncurrent smoker, and otherwise unremarkable subject was exposed to the approximately 75th percentile of each pollutant (e.g., 1.60 mg/m^{3}of PM). We excluded this subject from all analyses.

Firefighters spent an average of 5.4 ± 2.4 hours (70% of their time) exposed to some level of smoke between the time of

TABLE I. Pre- to Postshift TWA Concentrations for Pollution Variables

Variable	N	Mean	SD[A]	Minimum	Maximum
$PM_{3.5}$ (mg/m^3)	65	0.88	0.90	0.051	3.96
CO (ppm)	65	7.19	6.86	0.275	27.3
Formaldehyde (ppm)	65	0.054	0.060	0	0.21
Acrolein (ppm)	65	0.010	0.01	0	0.041
E_m (unitless)	65	0.58	0.59	0.027	2.34

[A] SD = standard deviation.

TABLE II. Pearson Correlations Among Pollutants

	$PM_{3.5}$	CO	Formaldehyde	Acrolein
$PM_{3.5}$	1			
CO	0.97	1		
Formaldehyde	0.96	0.97	1	
Acrolein	0.93	0.95	0.96	1

their preshift and postshift lung function tests. Cross-shift TWA concentrations for each pollutant are summarized in Table I. The pollutant concentrations are significantly higher than what are found in typical urban environments. Pearson correlations among pollutants are shown in Table II. All of the pollutants were highly correlated, which makes separating out individual effects of a single pollutant difficult.

An average of 7.6 ± 1.9 hours (range = 5.1, 13.2) elapsed between taking the preshift and the postshift lung function measurement. Lung function was found to be significantly decreased from pre- to postshift according to all lung function measures (Table III). These decrements remained significant even when exclusions for allergies, pre-existing lung conditions, smoking status, or recent colds were made, which is reported in more detail by Betchley et al.[7]

Using linear regression, we examined the association of $PM_{3.5}$, formaldehyde, acrolein, and CO with the pre- to postshift change in three lung function measurements: FEV_1, FVC, and FEF_{25-75}. No significant associations were found (Table IV). A 1000 $\mu g/m^3$ increase in $PM_{3.5}$ was associated with a –0.030 L change in the pre- to postshift change in FEV_1, a −0.024 L change in FVC, and a −0.11 L/min change in FEF_{25-75}. Each of these results is consistent with PM causing greater lung function decrements, but none of the results are statistically significant. Among the four pollutants, acrolein was most strongly related to pre- to postshift change in lung function.

We also fit multiple linear regression models to control for the effects of gender, age, and smoking status (Table IV). Adding these variables attenuated the association between each pollutant and lung function test. Gender, age, and being a current smoker were also not associated with larger or smaller FEV_1, FVC, or FEF_{25-75} decrements.

DISCUSSION

The earlier study with these firefighters[7] found that an 8-hr shift performing prescribed burns was associated with a significant pre- to postshift decrement in lung function values. However, our analysis did not find any significant difference in lung function decrements based on the component of exposure; that is, $PM_{3.5}$, CO, formaldehyde, or acrolein. Also, we could not detect a dose-response relationship for exposure to any of these pollutants. According to these data, there did not appear to be a larger decrement in lung function associated with exposure to 4000 $\mu g/m^3$ than to 51 $\mu g/m^3$ of $PM_{3.5}$. Thus, within our range of measured exposures, there does not appear to be strong evidence of a threshold value above which pollution exposure is particularly harmful.

These data suggest that a maximum lung response to fine particles occurs and that increasing the concentration of exposure does not lead to a stronger functional effect on the lung. At this point it is important to note that our only measure of adverse respiratory effect was spirometry. We did not design the study to incorporate measures of airway inflammation, such as a methacholine challenge or other noninvasive measures, or increases in cells collected by induced sputum or lung lavage. We attempted to control for current smoking status, gender, and age, but we may have had too few subjects to do so adequately.

This study has important implications for respiratory health of firefighters. Although the average percent decrease in FEV_1 from pre- to postshift was only 3%, average FEF_{25-75} decreased by 8%. Also, a one unit increase in each pollutant was associated with a larger cross-shift change in FEF_{25-75} than in FEV_1 or FVC. However, the association was not statistically stronger with FEF_{25-75}, partially because FEF_{25-75} is not measured as accurately as FEV_1 or FVC. FEF_{25-75} is thought to be a measure of smaller airway dysfunction and could suggest that the smoke exposure effects were predominately in the peripheral lung.

Rothman et al.[4] studied pulmonary function and respiratory symptoms in wildland firefighters in California looking at cross-seasonal effects. The cross-seasonal change in FEV_1 was –1.2% and for FVC was −0.3%. FEF_{25-75} was not measured. There was no air monitoring in their study; degree of exposure was estimated by hours spent fighting the fires. Cumulative hours of firefighting were associated with a decline in FEV_1 ($r^2 = .15$, $p = .006$). These data suggest that multiple-day exposures may not lead to greater lung function changes than single day exposures.

Another study undertaken to evaluate the cross-season effects of forest fire smoke on lung function and airway responsiveness reported significant mean declines in FEV_1 (−0.09 mL or 2%), FVC (−0.15 mL or 4%), and FEF_{25-75}

TABLE III. Mean Pre-Shift, Postshift, and Cross-Shift Change in FEV_1, FVC, and FEF_{25-75}

Lung Function Test	N	Preshift	Postshift	Change	95% CI for Change
FEV_1 (L)	63	4.43	4.31	−0.125	[−.176, −.074]
FVC (L)	65	5.21	5.14	−0.067	[−.107, −.026]
FEF_{25-75} (L/min)	65	5.27	4.82	−0.451	[−.652, −.249]

TABLE IV. Associations of Lung Function Measurements with Respiratory Pollutants

		Unadjusted		Adjusted[A]	
Lung Function Test	**Pollutant**	**Beta**	**p-Value**	**Beta**	**p-Value**
FEV_1	$PM_{3.5}$ (mg/m^3)	−0.030	0.28	−0.020	0.48
FEV_1	CO (ppm)	−0.0042	0.26	−0.0031	0.42
FEV_1	formaldehyde (ppm)	−0.42	0.33	−0.31	0.47
FEV_1	acrolein (ppm)	−3.30	0.21	−2.58	0.34
FEV_1	E_m	−0.047	0.28	−0.034	0.45
FVC	$PM_{3.5}$ (mg/m^3)	−0.024	0.30	−0.020	0.39
FVC	CO (ppm)	−0.004	0.24	−0.003	0.37
FVC	formaldehyde (ppm)	−0.36	0.30	−0.32	0.36
FVC	acrolein (ppm)	−2.73	0.20	−2.35	0.28
FVC	E_m	−0.039	0.27	−0.033	0.35
FEF_{25-75}	$PM_{3.5}$ (mg/m^3)	−0.11	0.32	−0.10	0.40
FEF_{25-75}	CO (ppm)	−0.015	0.31	−0.015	0.35
FEF_{25-75}	formaldehyde (ppm)	−1.35	0.43	−1.23	0.50
FEF_{25-75}	acrolein (ppm)	−16.6	0.12	−16.3	0.14
FEF_{25-75}	E_m	−0.18	0.30	−0.167	0.36

Note: Slopes are for a 1 mg/m^3 increase in $PM_{3.5}$, a 1 ppm increase in CO, formaldehyde, or acrolein, and a 1 unit increase in E_m.
[A]Adjusted for age, gender, and current smoking status.

(−0.44 mL or 11%) in 69 subjects.[5] These results agree with the present study in that the largest effect was seen in FEF_{25-75}. The Liu study[5] also reported a significant increase in airway hyperreponsiveness as measured by a methacholine challenge test ($p = .02$).

Exposure data for these subjects was reported separately in an industrial hygiene report.[14] They monitored exposure to CO, total and respirable PM, polyaromatic hydrocarbons (PAHs), silica, aldehydes, and benzene. CO concentrations were given for different fire fighting job categories and were in the same range as our study, for example, during the post-flaming phase of the fires (mop-up), CO ranged from 3–25 ppm. Respirable $PM_{3.5}$ during mop-up ranged from 0.327–5.14 mg/m^3, and formaldehyde ranged from 0.048 to 0.42 ppm. Acrolein samples could not be quantified; however, the highest value was estimated to be 0.023 mg/m^3.

Effects of community exposure to smoke for wildland forest fires have been reported in two studies. Duclos et al.[15] tallied emergency department visits for asthma and chronic obstructive pulmonary disease (COPD) from 15 area hospitals during a period of major forest fires in California in 1987. Significantly more patients visited hospitals during the forest fire period compared with a control period (observed/expected ratio = 1.4 for asthma and 1.3 for COPD), but no monitoring data was included. The fires destroyed in excess of 600,000 acres of forest.

Visits to emergency departments during a forest fire episode in Florida in 1998 were compared to similar visits for the same time period (June 1–July 6) in 1997.[16] Results showed a 91% increase in visits for asthma, 37% increase for chest pain, and 32% increase for shortness of breath. Of the 13 diagnoses compared, only 3 showed a decrease from rates in 1997. Those were for bronchitis (−20%), painful respiration (−27%), and palpitations (−21%).

The community data suggest that susceptible populations such as those with asthma show a much larger adverse effect from exposure to forest fire smoke than do the firefighters.[3,16] The relatively small lung function decrements seen in our study probably reflect, in part, the healthy worker effect. This is plausible because wildland firefighters are generally very fit individuals, accustomed to high levels of exertion. There was some evidence that increased exposure to $PM_{3.5}$, CO, acrolein, and formaldehyde led to greater lung function decrements, but a definitive association was not found in this group of firefighters. Another possible explanation for these results is that the threshold for pulmonary function effects of fine PM is low, perhaps in the range of 30–110 $\mu g/m^3$ as cited by the U.S. Environmental Protection Agency.[3] That may explain the lack of an exposure-response relationship in these data.

CONCLUSION

We conclude that the prescribed burn smoke exposures were associated with significant pre- to postshift decrements in lung function, as stated in a previous publication.[7] Our analysis was not able to identify any single component of the smoke that was significantly associated with the lung decrements. Identification of the effects of single toxic components in complex mixtures is fraught with great difficulty. In this light, our study indicates more research is needed to determine further the health effects of prescribed fire smoke exposure to firefighters. It may be necessary to develop more sensitive measures of appropriate health endpoints to achieve this goal.

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